

TELE-REHABILITATION GUIDELINE

Neurogenic Bladder Management in Spinal Cord Injuries

Drafted: 2014

Peer Reviewed:

Finalized: June 2014

70-84% of people with SCI will have some level of bladder dysfunction with a risk of upper and lower urinary tract complications (1). Therefore, formulating an individualized comprehensive bladder retraining and management program is essential. The goals are to preserve the upper urinary tracts, minimize lower urinary tract complications and minimize incontinence.

I. Neuroanatomy of the lower urinary tract

A. Innervation

1. The bladder receives innervation from both parasympathetic and sympathetic nervous system.
2. Sympathetic innervations (SI) to the lower urinary tract arise from the T11-L2 cord level.
3. SI synapses are located in the inferior mesenteric and hypogastric plexuses, and innervation courses through the hypogastric nerve.
4. Activation of the SI produces norepinephrine to be released in the lower urinary tract, resulting in detrusor relaxation, bladder neck contraction and internal sphincter contraction.
5. Parasympathetic innervation (PI) to the lower urinary tract arises from the S2-S4 cord level and is located within the pudendal nucleus.
6. Activation of the PI results in the release of acetylcholine and nitric oxide in the lower urinary tract, causing detrusor contraction and relaxation of the proximal urethra.
7. Somatic nervous system innervation to the external urethral sphincter arises from the pudendal nucleus at the S2-S4 cord level, which courses through the pudendal nerve to the sphincter striated muscle.
8. The majority of sensory afferent fibers are small myelinated A fibers and unmyelinated C fibers.
9. The A fibers have a graded response to bladder distention, while C fibers detect painful stimuli, temperature and chemical stimuli.

B. Voiding centers

1. Normal micturition necessitates: the proper function of both the bladder and urethra; a detrusor of normal compliance; a physiologically competent urethral sphincter, facilitating a passive low pressure filling of the bladder; and coordination of detrusor contraction with internal and external urinary sphincter relaxation, enabling adequate voiding of the bladder.
2. The pontine micturition center (PMC) modulates the opposing effects of the parasympathetic and sympathetic nervous systems on the lower urinary tract.
3. In the emptying stage, the PMC sends excitatory influence to the sacral spinal cord, producing detrusor contraction while simultaneously sending inhibitory influence to the thoracolumbar cord to relax the internal sphincter.
4. PMC inhibition causes suppression of the sacral spinal cord, resulting in detrusor relaxation. Simultaneously, thoracolumbar cord activation produces internal urethral sphincter contraction.

5. The sacral micturition center (SMC), at the level of S2–S4, is primarily a reflex center in which efferent parasympathetic impulses to the bladder cause bladder contraction, and afferent impulses to the sacral micturition center to provide feedback regarding bladder fullness.
6. Supraspinal centers, which normally are under voluntary control, produce excitatory influence on the pudendal nucleus during the bladder filling stage to produce external urethral sphincter and pelvic floor contraction to help maintain continence. During the bladder voiding stage, this descending influence is inhibited to produce urethral and pelvic floor relaxation to facilitate bladder emptying.

II. Pathophysiology and classification of neurogenic bladder

- A. Mechanical or physiologic defects in the bladder could result in an inability to regulate filling pressure.
- B. Neurogenic bladder is the loss of normal bladder function caused by damage to part of the nervous system.
- C. The damage can cause the bladder to be underactive, resulting in loss of contraction and complete emptying.
- D. The damage can also cause the bladder to be overactive, resulting in frequent and uninhibited contractions, with loss of synergy in bladder contractions and sphincter relaxation.
- E. Neurogenic bladder can be classified based on different criteria. The following classification is mainly formulated based on the level of injury in the central nerves system and helps guide pharmacologic and surgical intervention.
 1. Uninhibited bladder
 - a. Lesion is above the pontine micturition center (i.e. stroke, brain injury, or brain tumor).
 - b. There is reduced awareness of bladder fullness with a low pressure filling system, resulting in a low bladder capacity.
 - c. Since the PMC is intact, the normal opposition of detrusor and internal/external sphincter tonus is maintained and does not put the upper urinary tract at risk of high pressure damage.
 2. Upper motor neuron bladder
 - a. Lesion is between the pontine micturition center and sacral spinal cord (i.e. traumatic spinal cord injury or multiple sclerosis).
 - b. The spinal cord damage renders the bladder and sphincters spastic, especially if lesions are above the T10 level.
 - c. It is characterized by detrusor sphincter dyssynergia (DSD), where simultaneous detrusor and urinary sphincter contractions create a high pressure system.
 - d. Over time, DSD can lead to vesicoureteral reflux and renal damage.
 - e. The bladder capacity is usually reduced due to the high detrusor tone and loss of compliance.
 3. Lower motor neuron bladder
 - a. Lesion is at the level of sacral cord (i.e. traumatic spinal cord injury, multiple sclerosis or sacral nerve root injuries).
 - b. The SMC or related peripheral nerves are damaged while the sympathetic outflow is usually intact, resulting in a large capacity bladder since innervation to the internal sphincter remains intact and low detrusor tone bladder, associated with overflow urinary incontinence.

III. Evaluations

- A. Detailed history and physical examination is essential for accurate diagnosis and appropriate management.
- B. Neurological examination should include mental status, reflexes, strength, and sensation, including sacral dermatomes, to determine if there are neurologic conditions present that may contribute to the voiding dysfunction.
- C. Detailed genitourinary history needs to be taken.
- D. Medication history is also essential to eliminate any reversible causes.
- E. Urologic examination will need to include
 1. Urinalysis with culture and sensitivity (UA/c&s), Blood Urea Nitrogen/Creatinine ratio (BUN/Cr) and kidney functions
 2. Post void residual (PVR)
 3. Renal scan and/or Renal ultrasound
 4. Urodynamic evaluation (definitive and objective measure of bladder function)
 5. Cystogram/Cystoscopy
- F. Abnormal residual volume is 100 ml or greater than 20% of the voided volume, and residual urine volumes under 100 ml are associated with a reduced risk of development of bacterial cystitis.(6)
- G. Urodynamic evaluation should be completed to assess urinary function, including urinary flowmetry, bladder cystometrogram/electromyogram (CMG/EMG), Valsalva leak point pressure (LPP) measurement, and urethral pressure profile (UPP).
- H. Urodynamic studies are the most definitive and objective means to determine abnormalities in the bladder and urethra in the filling/storage phase and voiding phase in neurogenic bladder dysfunction.
- I. Normal filling pressure is 40 cm of H₂O or less.(11)

IV. Goals of management

- A. Achieve/maintain continence
- B. Prevent development of a high pressure detrusor that can lead to upper urinary tract damage
- C. Minimize symptomatic urinary tract infections and over-distension of the bladder

V. Nonpharmacologic intervention

- A. Intermittent catheterization
 1. Requires sufficient hand skills or willing care giver
 2. Needs to be avoided in patients with abnormal urethral anatomy, bladder capacity less than 200 ml, poor cognition/motivation or high fluid intake
 3. May require fluid schedule and restrictions (1800 ml/day, 400 cc with meals, 200 cc TID)
 4. Complications: UTI, urethral trauma, incontinence with over distention.
- B. Credé and Valsalva
 1. For individuals with lower motor injury with low outlet resistance.
 2. Avoid in patients with detrusor sphincter dyssynergia, bladder outlet obstruction, vesicoureteral reflux or hydronephrosis.
 3. Complications: incomplete bladder emptying, high intra-vesical pressure, vesicoureteral reflux, hydronephrosis, abdominal bruising, possible hernia, pelvic organ prolapse or hemorrhoids.
- C. Indwelling urethral catheterization

1. For patients with poor hand skills, high fluid intake, cognitive impairment, active substance abuse, elevated detrusor pressures, lack of success with other less invasive bladder management methods.
 2. Complications: bladder stones, kidney stones, urethral erosions, epididymitis, recurrent symptomatic urinary tract infections, incontinence, pyelonephritis/hydronephrosis from bladder wall thickening, fibrosis or bladder cancer.
- D. Suprapubic catheterization: For patients with urethral abnormalities, stricture, false passages, bladder neck obstruction, urethral fistula, urethral discomfort, perineal skin breakdown as secondary to urine leakage/urethral incompetence, personal preference, desire to improve sexual genital function, prostatitis, urethritis or epididymo-orchitis
- E. Reflex voiding
1. For male patients who, post-spinal shock, demonstrate adequate bladder contractions and adequate hand skills or have a willing care giver to change a condom catheter.
 2. Requires a periodic urodynamic study to monitor bladder pressures.
- F. Timed voiding: For patient with uninhibited bladder and a low pressure system

VI. Pharmacologic interventions

- A. Alpha-1 Adrenergic Antagonists
1. Peripheral postsynaptic blockade of alpha-adrenergic receptors in the bladder neck and proximal urethra to reduce urinary outflow resistance.
 2. Dibenzyline, terazosin, tamsulosin, alfuzosin, and doxazosin
 3. Reduction in blood pressure due to vasodilating effect on arterial smooth muscle.
 4. Side effects include fatigue, dizziness, lightheadedness, dry mouth and constipation.
 5. Can be used in conjunction with transurethral sphincterotomy.
- B. Anticholinergic (antimuscarinic) medications reduce reflex (involuntary) detrusor activity by blocking cholinergic transmission at muscarinic receptors and are the first-line option for treating neurogenic detrusor overactivity. (8)
1. Non-selective
 - a. Oxybutynin (Ditropan), tolterodine (Detrol LA), and trospium chloride
 - b. Side effect includes QT interval prolongation and memory and cognition impairments.
 2. Selective for M2 and M3 receptors
 - a. Solifenacin (Vesicare) and darifenacin (Enablex)
 - b. Fewer cognitive side effects
- C. Botulinum toxin (7,9)
1. Blocks neuromuscular junction presynaptic vesicle fusion and prevents acetylcholine release
 2. Acts on sensory afferent neurons and prevents the excitatory effects of nerve growth factor; effects can last for up to nine months
 3. Invasive procedure with intramuscular injections.
- D. Antibiotic medications should be used only to treat symptomatic urinary tract infections (UTI).
1. An UTI may manifest differently in patients with SCI than in the general population. The complaints of dysuria, frequency, and urgency are usually absent in infected patients with SCI (4). Common manifestations of UTI in patients with SCI include unusual fatigue, worsening muscle spasms, increasing autonomic dysreflexia, **urinary** leakage, and change in voiding habits. Fever is usually, but not always, present.

2. The definition of symptomatic UTI in these patients requires the presence of significant bacteriuria ($\geq 10^5$ CFU/ml); pyuria ($> 10^4$ WBC/ml of uncentrifuged urine or > 10 WBC/hpf for spun urine); and fever ($> 100^\circ\text{F}$) plus more than one of the following signs and symptoms: suprapubic or flank discomfort, bladder spasm, change in voiding habits, increased spasticity, and worsening dysreflexia, provided that no other potential etiologies for these clinical manifestations are identified. (5)
3. Most cases of bacteriuria in patients with SCI represent asymptomatic bladder colonization. Although asymptomatic bladder colonization may progress to symptomatic **infection**, often it does not. (2, 13)

VII. Procedure to enhance detrusor storage

- A. Enterocystoplasty increases bladder capacity(12)
- B. This procedure creates an anastomosis to join a part of the ilium or ileocecal segment to the detrusor
- C. High complication rate post-surgery, mucus production which can clog catheters
- D. Myomyotomy or myomectomy creates a large detrusor diverticulum

VIII. Procedures to control detrusor emptying

- A. Unilateral or bilateral sacral nerve root stimulation
- B. Dorsal rhizotomy
- C. Urinary diversion
- D. Continent abdominal stoma for clean intermittent catheterization (CIC)
- E. Appendicovesicostomy anastomosis

IX. Bladder sphincter procedures to enhance emptying

- A. Sphincterotomy
- B. Internal/external sphincter resection
- C. Urethral stents/balloon dilatation
- D. Artificial urinary sphincter devices
- E. Botox injections into the internal/external sphincter muscles.

This guideline was developed to improve health care access in Arkansas and to aid health care providers in making decisions about appropriate patient care. The needs of the individual patient, resources available, and limitations unique to the institution or type of practice may warrant variations.

Guideline Developers

Guideline developed by Noadia Worku, D.O in collaboration with the TRIUMPH team led by Thomas Kiser, MD, and Rani Lindberg, MD.

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